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Esophageal Ulceration Caused by Wax-Matrix Potassium Chloride

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THE INGESTION of enteric coated potassium chloride tablets to prevent potassium depletion during diuretic therapy has been associated with the occurrence of intestinal ulceration and gastric ulceration.[1-3] There have been recent reports of esophageal ulceration occurring in five patients[4-8] and esophageal stricture occurring in five patients[8-9] following therapy with orally given potassium chloride. In nine of the ten cases, the patients were taking tablets in which the potassium chloride was incorporated into a wax-matrix (Slow-K®),[4-6,8,9] while in the tenth case the patient was taking noncoated potassium chloride tablets.[7] The present case and a review of the other case reports suggest that the mechanism of esophageal ulceration is related to a narrowing of the esophagus usually due to an enlarged heart.

Report of a Case

A 49-year-old white woman with a history of chronic congestive heart failure was admitted to Memorial Hospital, Long Beach, California, on April 29, 1976. She had complaint of cough, shortness of breath, nausea, vomiting, abdominal pain and fever. An electrocardiogram showed left atrial enlargement and an x-ray study of the chest showed a right pleural effusion, and gross cardiomegaly with predominant left ventricular enlargement. A pulmonary angiogram showed a large clot present in a right lower lobe pulmonary artery. Initial anticoagulation was carried out with heparin followed by warfarin sodium (Coumadin®). Other medications included digoxin, furosemide (Lasix®), quinidine gluconate (Quinaglute®), spironolactone (Aldactone®) and potassium chloride liquid. Some heartburn and dysphagia developed on May 22 but improved over the next few days. On May 27 the potassium chloride liquid was discontinued and therapy with Slow-K (two tablets a day) was started. She took two tablets on May 27 but refused to take any more the following two days because of increased difficulty and pain on swallowing. She was discharged on May 29 in a stable but poor condition, being extremely dyspneic with cardiomegaly and chronic congestive heart failure. Her discharge medications included digoxin, quinidine gluconate, warfarin, spironolactone, furosemide and Slow-K (two tablets per day).

She was readmitted to the hospital on June 3, 1976, because of an almost total inability to swallow any food because of pain. Weakness, increasing shortness of breath and confusion were also noted. The admitting diagnosis was cardiomyopathy with congestive heart failure and dysphagia of unknown cause. In spite of increasing pain and difficulty on swallowing, she had continued to take her medications including her Slow-K tablets since her discharge five days previously. The Slow-K was stopped on admission. An electrocardiogram showed a left bundle branch block, inferior lateral fibrosis and left atrial enlargement. An x-ray film of the chest showed congestive heart failure, cardiomegaly and bilateral pleural effusions. An esophagram done on June 4, 1976, showed a polypoid mass measuring approximately 6.5 cm in longitudinal dimension and located at the level of the 6th, 7th and 8th thoracic vertebrae immediately behind the point of maximal compression by the enlarged left ventricle. An upper gastrointestinal study done on April 26, 1976, had shown a normal esophagus with no lesion present. The differential diagnosis of the mass included carcinoma of the esophagus or an ulcerating inflammatory lesion with edematous and inflammatory tissue. Endoscopy was done on June 7 and showed a midesophageal constricting necrotic lesion and esophageal biopsy specimens were obtained. The biopsy findings were reported as benign squamous mucosa and fibrinopurulent exudate consistent with a benign esophageal ulcer. Over the next week and a half the patient noted decreased pain and difficulty in swallowing. A repeat esophagram on June 14 showed an ulcerated mucosa of the esophagus at the level of the 6th, 7th and 8th thoracic vertebrae, the intraluminal component of which had decreased in size since the previous examination. She noted further

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gradual improvement in swallowing and had no further pain when she was discharged on June 20, 1976.

Discussion

Five cases of esophageal ulceration associated with potassium chloride therapy have been reported in the literature. This is the sixth reported case. These six patients and the five additional patients in whom esophageal stricture developed secondary to potassium chloride therapy ranged in age from 33 to 66 years. Nine were women and two were men. Ten of these patients had taken Slow-K. The dosage, when stated, ranged from 1,200 to 3,600 mg per day. The duration of therapy in these patients is not always clearly stated in the case reports but would appear to range from six days (in the present case) to several months of therapy.

A probable predisposing factor in 9 of the 11 cases appeared to be esophageal compression secondary to left atrial enlargement. In four of the patients esophageal ulceration developed[5-8] and in five esophageal stricture developed following cardiac surgical operation.[8,9] In the other two cases of esophageal ulceration, including the present case, cardiac surgical procedures had not been done. The present patient had left ventricular enlargement and cardiomyopathy.

Compression of the esophagus due to cardiac enlargement may result in stasis and dilatation of the esophagus. Clinical and experimental evidence indicates that the local effect of the hypertonic potassium chloride combined with a relative vascular insufficiency is associated with ulceration in the small bowel.[2,10]

Esophageal ulceration secondary to the administration of wax-matrix potassium chloride or noncoated potassium chloride tablets is a potentially serious complication. Death has resulted in three cases. In two patients, cause of death was hemorrhage.[4,8] The other death was attributed to sepsis related to the septic process in and around the esophageal ulcer.[7] Two other patients required feeding via a jejunostomy and later died.[8]

Slow-K induced esophageal ulceration is a potentially serious complication that may develop in patients with esophageal compression. Patients with cardiac enlargement which may result in esophageal compression should not be given potassium chloride tablets. These tablets should also be avoided in patients with esophageal compression due to other causes.

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Vasculitis Associated With Propylthiouracil

Evidence for Immune Complex Pathogenesis and Response to Therapy

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PROPYLTHIOURACIL - INDUCED VASCULITIS has been reported previously.[1-6] McCormick[1] established the hypersensitivity nature of the syndrome by showing that the disease recurred when the drug was reintroduced. Inflammatory changes in blood vessels and of skin, liver, central nervous system, pancreas and kidney have been described.

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